Thus, at length, we decided to provide a simplified list here of ways to assure renewal and obtainment of methylene bridges that are essential to regenerative repair and indefinitely sustainable wellness along with downregulation of elevated methylene bridge cysteine while downregulating methylene bridge cysteine to decrease this required factor in detrimental aspects of aging and disease.

Alleviating Methylene Bridge Cysteine and assuring Methylene Bridge availability otherwise

Objectives in this regard to is to manage methylene bridge cysteine, or eHCY, to below 6 or7 um/L with a objective of near 3.7 um/L. Methylene bridge pathways can begin with ethanolamine in the CDP-ethanolamine pathway, where PEMT packs methyl groups into phosphatidylethanolamine to produce hydride/methyl enriched phosphatidylcholine and resolution phase cytokine substrate enriched phosphatidylcholine. While methylene bridges enable DNA, RNA, NADP+/NADPH, Fatty acid, alkane or other essential polymerizations to occur, while also performing as antihistamines, autophagy promoters, and performing as and enabling synthesis of primary phospholipids of cellular physiology. The primary incipient factors in disease and detrimental aspects of aging include cellular existentialism challenges from inadequate enriched phosphatidylethanolamine and enriched phosphatidylcholine as well as the functions of antihistamines in the CDP-ethanolamine/PEMT pathways.

The factors here provide topical, ingestible, nutritional, clinical, mechanism that can be customized circumstantially to alleviate detrimental aspects of aging, enable regenerative repair, ameliorate disease of movement, cognition, cardiovascular plasticity, and impaired plasticity.

These factors can assist other therapies or replace some other therapies.

DHA enriched Phosphatidylethanolamine, phosphatidylserine, Ca2+ and Ca2+ as part of a mineral supplement also, utilize diverse hexose sugars instead of glucose to circumvent P53 blockade of glute1/3/4 endocytosis of sugar that occurs when PEMT is downregulate or injury causes P53 upregulation, Vitamin K2 to alleviate Ca2+ dysregulation, pomegranate extract for mitochondrial resilience, Phosphatidylinositol, collagen, laminin, DHA Enriched Phosphatidylcholine, Methyl shift, Hydride Shift, BHMT, BHMT2, Methione Synthase, s-methylmethionine sulfonium, methylsulfonylmethiane, danshen/redsage/salviaM for transsulfuration pathway activation, INMT, THMT, s adenosyl methionine, Active Hexose Correlated Compound and a Choline Kinase inhibitor, Curcumin and SP1 downregulator with PD1/PDL1 downregulator, Berberine and a AP1 downregulator, phosphatidylethanolamine, phosphatidylcholine, Selenium (trimethylselenonium, selenium conjugates such as Methylselenol or Methylselenic Acid) CRISPR CAS9 gene repair using transduction domains

for complete coverage of all cellular entities, EMF protection, Filtered water, trimethylsulfonium, Tetrahydryofolate methyltransferase obtainable as gastromend hp of BHMT2 activation, PEMT, B6, B12, Selenonium, NOPE1, NOPE2, 1-palmitoyl-2-oleoyl-phosphatidiylethanolamine, NAPE, anandamide and other n-acyl ethanolamines including PEA, OEA, NAE, AEA, DHEA/synaptamide/n-docosahexaenoylethanolamine, n-acetylethanolamine, complete mineral supplement with potassium/magnesium, 2 pinches of ancient pink Himalayan sea salt, DHA, EPA, Oleoylate, Trimethylglycine/Betaine/glycinebetaine for BHMT1 activation, 6s 5678 tetrahydrofolate, Cystathionine for CBS pathway, n-palmitoylethanolamine, n-oleoylethanolamine, n-arachidonoylethanolamine, Nad+/Nadh supplement, Hydrogen Supplement, dimethylactothetin for desquamation and Cysteine bridge depletion, Nitric Oxide Donor, Math+/HAT therapy, N-acetyl L cysteine, Indole 2 3 2oxygenase downregulator such as moringa oliefera or Moringa Tea, kolaviron or kola berry extract, Methylene Blue or Methylene blue and phototherapy, iNOS inhbitor or curcumin/irinotecan Lname/Lnmma, L-arginine, L-citrulline, L-ornithine, activated vitamin D, Ca2+, , Enlyte/EnlyteRX, topical thioglycolic acid, 26 s proteosome inhibitor, 19 S proteosome inhibitor, BAG1 inhibitor, whole animal glandular supplement for agrin and matrix content, agrin grafts to cardiac and other tissue, laminin granules as designer proteins or granularized connective tissue/matrix from food organisms, granularized connective tissue/matrix, kidney stuff by golden standards, TMAO downregulation and Carotid Intima Media flow/plasticity assurance and leaky gut downregulation with 33 dmb/tmaLyasedownregulator/RegularLaxative/Prebiotic/Probiotic(especially BifidoInfantis)/Postbiotic/Macrobiotic/BroadSpectrumAntibioticforEmergency/OliveOil/GrapeseedOil, A vitamin supplement that includes maximum levels of diverse plants/oils/sources, peroxiredoxin, glutathione, reduced glutathione, dimethylsulfide, Ethanolamine, molecular hydrogen, therapeutic nitric oxide, nose breathing to enhance nitric oxide production, S1P receptor down regulators, GSK3B inhibitor or Curcumin/watercress, Monocyte chemoattractant Protein1 downregulator or curcumin, GCPR receptor down regulators, bone powder along with methylsulfonyl methane also with agrin and also with whole organism glandular along with phosphatidylcholine for bone and marrow pathology, short duration nonstrenuous exercise using resistance, RNA/DNA nucleotide supplement, Ribose, Dimethylacetothetin to diminish methylene bridge cysteine, only ingest meat/chicken/eggs/fish if they are micronized/granularized and are ingested with probiotic/prebiotic/postbiotic/macrobiotic/oliveoil/balsamicvinager/grapeseedoil to prevent TMAO, always monitor for and counteract for L lactate/L lactic acid and D lactate/D lactic acid, orexins are factors that elute consciousness often assisted by stimuli in the environment that perform as a frame to keep conscious focus and sustain arousal.

EMF protection should be applied to a whole area, whole anatomy, and particularly above, below and around an area of physiology exhibiting pathology.

Additional Information

Managing methylene bridge cysteine or the clinical indicator eHCY or elevated Hcy

Elevated methylene bridge cysteine or elevated HCY recommended to be managed toward 3.7 um/L may be optimal. Above 6 or 7 um/L risk increases 99.995% requirement at least managed therapy. 10 um/L or higher requires inpatient therapy if accompanied by symptoms and outpatient/office therapy if not accompanied by systems. 15 um/L or higher require inpatient therapy. Methylene bridge cysteine treatment toward 3.7 um/L should be a priority in medical, physiological, regenerative, behavioral, emergency, acute, subacute, inpatient, outpatient, office, home, allopathic, traditional, Eastern and Wholistic care. S-adenosyl methylene bridge cysteine should be therapeutically targeted for 0.012 um/L.

1) Elevated HCY, clinical indicator HCY or eHCY, methylene bridge cysteine
    a) Bystolic or Nebivolol. Saline. NMDA Receptor inhibitors
    b) Phosphatidylcholine, Choline, Alpha-GPC, Choline Kinase alpha inhibitor Pregnenolone, DHEA, S - Methylmethionine sulfonium, Methylsulfonylmethane, A complete mineral supplements, minerals from pink Himalayan sea salt, a complete natural vitamin supplement with B12/B6/thiamine/pantothenic acid/K2/Biotin, Riboflavin, other vitamins. Glutathione. Catalase. Selenium. Sulfobetaine. Superoxide Dismutase. N Acetyl L Cysteine. Peroxiredoxin-6. Cysteine. Histidine. Cystathionine.
2) Transsulfuration Pathway Depletion of methylene bridges to nonmethylene bridge cysteine.
    a) This suggests that sulfur should be added to B6, Methionine, NAD+, Serine, Danshen/Red Sage/Salvia M, Propionate, Succinate.
    b) Metabolites Cystathionine, Cysteine, Alpha-Ketobutyrate, CoA, Glutathione, and simple Sulfates such as H2S or HS, and Cystine.
3) Managing methylene bridge cysteic Acid, Derivative of methylene bridge cysteine
    a) Saline along with Alkalinization Therapy.
    b) Vitamin K1 and Vitamin K2 as Menaquione-4.
    c) NMDA Receptor inhibitors
4) Managing methylene bridge thiolactone, Derivative of methylene bridge cysteine
    a) However, PON1 by a number of factors.
    b) PON1 Translocation through SREBP2 and SP1 integration at the PON1 promoter occurs resultant of Statin, Quercetin and Glucose.
    c) PON1 activation through the aryl hydrocarbon receptor occurs resultant of Quercetin, Resveratrol and Aspirin utilization.
    d) Berberine, however, induces PON1 through the JNK-c-JUN signaling pathway. Resveratrol is a phytoalexin. trans 3,4,5,4′-tetramethoxystilbene
    e) Pomegranate juice polyphenolics stimulate PON1 expression through the PPARy-PKA-cAMP signaling pathway.
    f) Unknown mechanisms of action enable PON1 upregulation resultant of utilizing Curcumin, Betanin, Isothiocyanates, Licorice Polyphenolics, and olive oil.
5) BHMT Pathway for decreasing methylene bridge cysteine through recycling into Methionine

a) Glutathione. Trimethylglycine. 6s 5678 Tetrahydrofolate, Zinc. N Acetyl-L Cysteine, Peroxiredoxin.

6) BHMT2 Pathway methylene bridge cysteine through recycling into Methionine
    a) Glutathione. S-Methylmethionine (S – Methylmethionine Sulfonium). 6s 5678 Tetrahydrofolate, Zinc. N Acetyl-L Cysteine, Peroxiredoxin.
7) Thetin-methylene bridge cysteine Pathways decreasing methylene bridge cysteine or eHCY through recycling into Methionine
    a) Dimethylthetin, Trimethylsulfonium, dimethylsulfonioacetate, ethylmethylthetin, dimethyl-alpha-propiothetin, dimethyl-beta-propiothetin, ethyl methyl-beta-propiothetin, dimethyl-gamma-butyrothetin, methionine, methylsulfonium, trimethylsulfonium, ethyldimethylsulfonium, butyldimethylsulfonium.
8) Thiopurine/Thioether S – Methyltransferase
    a) Existing S-Adenosyl l methylene bridge cysteine , H+, and 6 methylthiopurine.
    b) 6 – methyl thioguanine, H+ and existing S -adenosyl L methylene bridge cysteine.
    c) Existing S -adenosyl L methylene bridge cysteine and a thiopurine s – methylether
9) Methionine Synthase
    a) 5, Methyltetrahydrofolate, Vitamin B12 Methylcobalamin
10) Trimethylsulfonium Tetrahydrofolate N Methyltransferase
    a) Trimethylsulfonium and 6s 5678 Tetrahydrofolate bidirectionally potentiates dimethylsulfide and 5 methyltetrahydrofolate
11) S-adenosyl Methionine Synthetase
    a) Methionine, Water and ATP, potentiate phosphate, diphosphate and S-Adenosyl Methionine.
12) MARS1/MARS2 Methionyl – tRNA – Methionyl Ligase
    a) Methionine is important because it is a starting factor or primer in synthesis of more than 99.5 percent of gene transcription products. MARS1, for instance, as Methionine tRNA Ligase catalyzes synthesis of AMP, diphosphate, L-methionyl tRNAMet from ATP, L – methionine and tRNAMet. MARS1 occurs in the Nucleus of Homo Sapiens and MARS2 occurs in the mitochondria, performing a role in enabling incipient nuances of synthesis of RNA in Ribosomal Molecular Machines.
13) S-adenosyl methylene bridge cysteine Hydrolase, SAH, SAHH
    a) NAD+ availability, compared to NADH, potentiates production of methylene bridge cysteine or HCY from S-Adenosyl methylene bridge cysteine or SAH.
14) INMT, Indolethylamine N – Methyltransferase, Thioether S - Methyltransferase
    a) Dimethyl Sulfide, Trimethylsulfonium, a primary methylated amine, a secondary methylated amine. 2-methylthioethanol, Dimethyl Selenide, Dimethyl Telluride, Diethylsulfide, Tryptamine, Diethylsulfide, all along with H+. Increased levels of S-Adenosyl Methionine can naturally potentiate this enzyme toward S-Adenosyl Methionine, but the trimethylated versions of these substrate are exclusive in catalyzing activity toward S –Adenosyl Methionine. Trimethylsulfonium, Trimethylselenonium, Trimethyltellurium , and possibly Trimethylglycine, although Trimethylglycine can be used by BHMT to produce Methionine and Dimethylglycine. Trimethylsulfonium produces linear graphs of the depletion of S-Adenosyl methylene bridge cysteine or SAH because it is used by TTMT

toward 6s 5678 Tetrahydrofolate/Dimethylsulfide, used toward Thioglycolic Acid/Methionine by Thetin – methylene bridge cysteine Methylpherase, and used toward S-Adenosyl Methionine/Dimethyl Sulfide.